Hiroshima J. Med. Sci.
Vol. 60, No. 3, 67~69, September, 2011
HIJM 60−13

A Case of Idiopathic Intracranial Hypertension Related with Vesicoureteral Reflux

Cihangir AKGÜN[1], Gökmen A. TAŞKIN[2], Sinan AKBAYRAM[2], Avni KAYA[3,*], Hayrettin TEMEL[2], Sevil Arı YUCA[3] and Hüseyin ÇAKSEN[4]

1) Yüzüncü Yıl University, Faculty of Medicine, Departments of Pediatric Nephrology, Van, Turkey
2) Yüzüncü Yıl University, Faculty of Medicine, Departments of Pediatrics, Van, Turkey
3) Van Women and Children's Hospital, Department of Pediatrics, Van, Turkey
4) Yüzüncü Yıl University, Faculty of Medicine, Departments of Pediatric Neurology, Van, Turkey

ABSTRACT

Pseudotumor cerebri is idiopathic intracranial hypertension. The etiology of this syndrome has not been fully clarified. Excess cerebrospinal fluid production, scarcity of cerebrospinal fluid absorption, intracranial venous pressure elevation, increased intracranial blood volume are all thought to be responsible. The symptoms of the disease may be ordered according to prevalence as follows: headache due to increased intracranial pressure, blurred vision and diplopia. A thirteen-year-old female patient was brought in with complaints of headache, double and blurred vision. Systemic arterial hypertension (140/70 mmHg) was determined. Vesicoureteral reflux was detected as the hypertension etiology. In this article a rare pseudotumor cerebri case is presented secondary to vesicoureteral reflux which caused hypertension.

Key words: *Intracranial hypertension, Child, Headache*

Idiopathic intracranial hypertension (IIH) is a clinical syndrome which occurs due to the increment of intracranial pressure with cerebrospinal fluid (CSF), normal cellular and protein content, normal ventricule size and position[1,9]. The etiology of the syndrome is not exactly clear. Excess production, decreased absorption of CSF, high intracranial venous pressure or increment of intracranial blood volume have been reported to be the cause[3]. Increased intracranial pressure, diagnosed by measuring pressure above 20 cmH_2O, and papilledema accompany the syndrome[9]. However, it should be mentioned that papilledema is not necessary for the diagnosis[6]. Systemic arterial hypertension was reported in IIH cases at a rate of 14-32%, and in one study it was suggested that blood pressure was higher than in control groups. But it is not known whether this association is independent from obesity[5].

In this report a IIH case secondary to hypertension due to vesicoureteral reflux is presented as it is seen infrequently.

CASE REPORT

A 13-year-old girl was admitted to the hospital complaining of headache, diplopia and blurred vision. According to her recollection, her symptoms had been present for one and a half years. Headaches had occurred almost every day and were unresponsive to analgesics. Headaches at the bilateral upper part of the head usually lasted 1 or 2 hr but sometimes continued all day. Sometimes she woke up with a headache. Before the start of a headache no preceding symptoms like smell, light flashes or sound were present. The headaches did not increase with stress, position change, eating, chewing, coughing or sneezing. She never fainted or had a seizure. Last year she had diplopia periodically. While she was looking at a light it appeared to elongate, but she did not have sensitivity to light.

At physical examination her general appearance was well. She was aware, was conscious while arterial tension was 140/70 mmHg (>95 percen-

*Correspondence: Dr. Avni KAYA
Office address: Women and Children's Hospital, Department of Pediatrics, Van, Turkey
Tel: +90432-217-1983, Fax:+90432-215-0479, E-mail: avnikaya@gmail.com

tile/50-75 percentile) and other vital signs were within normal limits. In her neurologic examination no localized sign other than papilledema was determined. During visual field evaluation visual field loss at the nasal quadrant's periphery on the right side and bilateral diffuse sensitivity loss were observed. An enlarged blind spot and a 2-piece scotoma at the left side were observed, however, no hypertensive retinopathy sign was present. Other systemic examinations were normal. While she had no special feature in her medical history, her mother had a history of hypertension.

In the light of these findings, the patient was diagnosed as IIH secondary to hypertension, and to reduce intracranial pressure, acetazolamide and steroid therapy were administered. Repeated lumbar punctures, which may be employed to decrease intracranial pressure in cases of intracranial hypertension, were not performed in this patient. Her brain magnetic resonance imaging was normal. Cavernous sinus thrombosis was not considered. Therefore brain magnetic resonance angiography was not performed. Nifedipine was started on the patient for systemic hypertension and a salt-free diet was ordered. The patient's glomerular filtration rate was normal. Renin level was detected as >10 ng/ml/hr (normal: 0.5-5.9 ng/ml/hr).

Laboratory investigations, total blood count and biochemistry parameters were normal. CSF pressure was measured as 25 cm H_2O and leukocyte and lymphocyte was not seen at microscopy. CSF biochemistry values, hormone profile and catecholamine (vanillylmandelic acid, metanephrine, normetanephrine and 5-hydroxyindole acetic acid) excretion at 24-hr urine were also normal. Because of their involvement in the etiology of IIH, the patient was investigated but no positive sign was found for vitamin A disorder, hypothyroidism, hypoparathyroidism, Cushing's disease or pregnancy.

Radiologic studies, brain and orbital magnetic resonance imaging were normal and no significant paroxysmal activity was detected at electroencephalogram. In renal Doppler ultrasonogram, the dimensions of the left kidney were attenuated and the contours were lobulated. Renal parenchyme echogenicity was slightly increased (grade 1 renal parenchymal disease). The arcuate arteries of both kidneys were within normal limits. Tc-99m dimercaptosuccinic acid was performed. As a result of Tc-99m diethylenetriamine pentaacetic acid, the left kidney was found to function normally with middle-inferior calicial stasis. After these findings voiding sistourethragraphy was performed and bilateral grade III vesicoureteral reflux was detected. Her echocardiography findings were normal.

It was observed that the patient's headache and vision problems resolved prior to blood pressure regulating therapies. Submucosal matter injection was preferred for the treatment of vesicoureteral reflux and the patient was transferred to a hospital with a pediatric surgery department. The patient underwent successful submucosal injection of substances by the subureteric injection technique. At post-operative follow-up both symptoms and blood pressure were stable.

DISCUSSION

IIH is seen rarely and it was reported that among the United States population its annual incidence ratio was 0.9/100,000[10]. IIH may develop as a result of many diseases and use of many drugs. It is usually seen in young, obese women[2]. Our patient was also young but no obesity was present. This syndrome was first described in 1897 by Quincke and was named 'serous meningitis'[10].

The exact etiology of this syndrome is not well known and it was named in later years as 'idiopathic intracranial hypertension', 'hypertensive meningeal hydrops', 'pseudoabscess', 'toxic hydrocephaly' and 'otitic hydrocephaly'[7]. Noetzel and Rioux[8] reported two infants with pseudotumor cerebri associated with renal disease. The pathogenesis of increased intracranial pressure is unclear, but possibly vesicoureteral reflux leads to systemic hypertension and systemic hypertension leads to intracranial hypertension. The most common symptoms are headache, transient blindness, blurred vision and diplopia. All of these symptoms were present in our patient.

It was proposed that IIH, also defined as idiopathic, is triggered by some factors, even if the etiology of this syndrome is not known exactly. The most well-known of these are obesity,

pregnancy, menstrual disorders, Addison's disease, hypervitaminosis A or hypovitaminosis A, steroid treatment or cessation, tetracycline treatment, hypoparathyroidism and iron deficiency anemia[9]. No pathology was detected in our patient. With IIH cases, systemic arterial hypertension has been reported at a rate of 14-32%[5]. The cause of hypertension varies according to age; hypertension etiology at the patient's age group was 30-40% due to vesicoureteral reflux[7]. In our case, subureteric injection therapy was performed. The patient in post-operative follow-up showed stable blood pressure. The patient's other symptoms, including headache, diplopia and blurred vision were completely resolved.

The prime goal of management is discovery and treatment of the underlying cause. There are no randomized clinical trials to guide the treatment of IIH. An obese patient should be treated with a weight loss regimen, and if a drug is thought to be responsible, it should be discontinued. The initial lumbar tap following a CT or MRI scan is diagnostic and may be therapeutic. Several additional lumbar taps and the removal of sufficient CSF to reduce the opening pressure by 50% occasionally lead to resolution of the process. Acetazolamide, 10–30 mg/kg/day, and corticosteroids are effective for some patients. Rarely, a lumboperitoneal shunt or subtemporal decompression is necessary if the aforementioned approaches are unsuccessful and optic nerve disorder supervenes. Some facilities perform optic nerve sheath fenestration to prevent further visual deterioration[4]. In our case acetazolamide and steroid therapy reduced intracranial pressure, though the lumbar puncture was ineffective.

In conclusion, for a patient with headache and visual symptoms it should be kept in mind that such symptoms may be caused by intracranial hypertension, the etiology of which should be elucidated by a full physical examination as well as the use of diagnostic modalities..

(Received November 5, 2010)
(Accepted August 8, 2011)

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